THE ROLE OF THE ALLOXURIC BASES IN THE PRODUCTION OF THE CARDIO-VASCULAR CHANGES OF NEPHRITIS.

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Hypertrophy of one or both ventricles, with or without dilatation, and increased arterial pressure are found in many cases of nephritis and in various lesions of the kidneys associated with destruction of parenchyma.

Of the theories promulgated in explanation of this phenomenon only two merit serious consideration, viz., the mechanical or physical theory created by Traube[1] and the intoxication or retention theory originated by Bright.[2]

Traube's mechanical theory is readily disproved. He postulates that two factors in nephritis cause increase of arterial pressure with resulting hypertrophic changes in the heart and bloodvessels, viz.: 1. The obliteration of numerous bloodvessels in the contracted kidney, parenchyma, or their compression by exudate in acute inflammation. 2. The increase of the quantity of fluid in the arteries as a result of the decreased abstraction of water by the reduced or diseased kidney parenchyma.

These premises are fallacious: First, because increased resistance in the renal arteries does not produce increased blood pressure, as has been conclusively demonstrated by Ludwig, who failed to obtain increased aortic pressure after ligation of both renal arteries. Second, because a diminished excretion of water with resulting plethora would not cause the aortic pressure to rise; experimental injections of physiological salt solution, causing an excess of fluid in the arteries, do not produce a rise of pressure, for the reason that the arteries equalize the blood pressure by their vasodilator mechanism. Third, because in that form of nephritis in which cardiac hypertrophy is most frequent, chronic (primary) interstitial nephritis, the excretion of water is not only not decreased, but, on the contrary, increased from the very beginning. Fourth, because the arteries are capable of getting rid of excessive water through other channels than the kidneys.

Bright's retention theory postulates that in nephritis certain excrementitious substances are poured into the circulation that by exercising either a direct cardio-excitory action cause hypertrophy of the heart, or by producing constriction of terminal arterioles and capillaries throw additional work on the heart muscle and thus cause enlargement.

(*a*) Arterio-sclerosis may be primary, the induration and atrophy of the kidneys secondary; " true sclerosis of the kidneys."

(*b*) As a result of parenchymatous nephritis with retention of excrementitious substances, both hypertrophy of the heart and arterio-sclerotic changes in the arteries may occur. In this case the nephritis is primary, the " arterio-sclerosis " secondary.

(*c*) The same toxins may simultaneously produce both the nephritis and the arterio-sclerosis (lead, gout, etc.).

Arterio-sclerosis is, therefore, only in isolated instances the cause of the nephritis. In the majority of instances, where the two conditions are found together, the arterio-sclerosis stands in no causal relationship to the nephritis, but is either a result or a concomitant feature. Where arterio-sclerosis is primary it usually constitutes a senile change. In all other forms it will be found that some kidney lesion coexists and that we are dealing with one of the forms described under (*b*) and (*c*).

Our investigations, therefore, justify us in attributing to the alloxuric bases an important rôle in the production of the cardio-vascular changes observed in *all* forms of nephritis with the exception of that chronic indurative form sometimes seen as a result of senile arterio-sclerosis.

A series of investigations, finally, instituted with varying doses of uric acid for different periods of time (see previous publication, Experiments I. to VIII.), revealed that uric acid is altogether without effect on the blood pressure, the arteries, and the heart.

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